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THE BRITISH JOURNAL
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MAY, 1943

COMMUNICATIONS

ON SOME ANOMALOUS FORMS OF AMAUROTIC IDIOCY AND THEIR BEARING ON THE RELATIONSHIP OF THE VARIOUS TYPES

BY

ROGER WYBURN-MASON, M.D.(Cantab.)

FROM THE NATIONAL HOSPITAL, QUEEN SQUARE

(Conclusion)

Anomalous Types

The occurrence of certain anomalous types has been doubted by some authors, *e.g.*, Sorsby, but the departures from the typical features are not uncommon. Mention will be made of some of these and examples given from personal cases.

A. INFANTILE TYPE.

1. *The presence of a cherry-red spot in other than infantile cases.*—Torrance in 1921, collected nine examples in which he considered a cherry-red spot had been present at the macula in juvenile cases. The description in most of these does not carry conviction, the appearances were not typical and were merely slight variations of the pigmentation usually occurring in the juvenile type, *e.g.*, cases of F. E. Batten, R. D. Batten, Kuffler, Ichikawa, and Rogalski. Gordon's cases were misdiagnoses for the Laurence-Moon-Biedl syndrome. Of the others, Wolfsohn's case, beginning

at three and a half years, showed at each macula a brownish-red spot surrounded by a greyish area with no other retinal changes. One of Condes and Horner's Japanese cases described as infantile, set in at one and a half years of age. In Higier's (1906) Jewish family, the fourth child had a typical cherry-red spot at the macula, when seen at the age of thirteen months. One of Liener and Goodhart's Jewish cases had its onset at the age of nine months. Greenfield and Nevin's non-Jewish case died shortly after being seen for the first time at the age of two years and ten months, and a red spot was present at the macula with a greyish area around it. In Case 7, the brother of their case, the first symptoms occurred at the age of ten months but the child did not die until three years later. The eldest patient in our L. family did not develop symptoms until the age of eighteen months although in two of the other siblings the onset of visual failure occurred before the age of one year. Case 10 in the Ha. family had no symptoms until the age of eight years and a typical cherry-red spot was present when seen two years later. She is by far the oldest patient in which such a sign has been recorded. The family are non-Jewish, there was no consanguinity and an elder sister died from the infantile type of the disease under the age of one year. The mother also showed mental symptoms, epilepsy and optic atrophy.

2. *Absence of the cherry-red spot.*—This occasionally occurs as in the cases of Hassin and Parmelee, Schlesinger *et al.*, Weber, Higier (1906) and the third case of the L. family. In all these cases the findings were those of a simple optic atrophy, but this may precede the development of a cherry-red spot as in Koller's case and it seems probable that had some of the cases been followed further, the typical cherry-red spot would have appeared. Further the appearance of the cherry-red spot may change as in Buchanan's case, where the red spot was surrounded by a greyish rather than a white zone. In Case 7, the brother of Greenfield and Nevin's case, the cherry-red spot was also surrounded by only a faint greyish zone.

Bertrand and V. Bogaert mention a family in which two infantile cases occurred and a third member showed dementia, the first symptoms occurring at the age of three. The patient was alive at the age of eighteen years, but the retinae were normal and this was thought to be a case of amaurotic idiocy without eye signs. It is probable that, just as in the brain, the severity of the changes varies from region to region, so in the retina, which may or may not be heavily involved.

3. *Occurrence among non-Jews.*—Slome, in 1933, found eighteen authentic non-Jewish cases among the two hundred or so reported. Examples are those of Cockayne and Attlee, Turner,

Levy, Magnus, Pooley, Muggia et al., Gil et al., Schlesinger et al., and Greenfield and Nevin (references will be found in the papers of Cohen and Condes and Horner). Seven further cases occurring in four further families are recorded here. (Cases 3-7 and 9-10).

4. *The onset of symptoms after the first year of life, and association of cases occurring in the infantile and late infantile periods in the same family.*—Typical examples of the former were found in the cases of Liener and Goodhart, Greenfield and Nevin, Higier (1906, fourth child), Condes and Horner, the first two patients of the L. family and in Case 10. Only the first and third of these cases was Jewish. A remarkable fact in some of these families is that some of the patients may be under the age of a year at the onset, *i.e.*, infantile, while in others the onset of symptoms is somewhat later and falls in the late infantile period. In addition some members of a family may show a typical cherry-red spot and others only optic atrophy, *e.g.*, Higier's (1906) Jewish family. In this family in which four children were affected the first and second cases began to lose their sight in the first year and only optic atrophy was present. In the third child no symptoms occurred until the fourth year and again only optic atrophy was present with mild imbecility. The fourth child was seen at the age of thirteen months and a cherry-red spot was found at the macula. This family cannot therefore be considered as an example of the association of juvenile and infantile cases as has been stated by previous authors, *e.g.*, Sorsby, but rather of cases with their onset at ages falling within the infantile and late infantile period. A similar association occurred in our J. family in which the elder patient (reported by Greenfield and Nevin) did not show symptoms until the third year of life while a younger brother (Case 7) began to deteriorate at the age of ten months. In our L. family, the first child had no symptoms until the age of eighteen months, the second until twelve months, both showing the typical cherry-red spot, while in the third visual failure began at five months of age and he died without the appearance of a cherry-red spot in the fundus.

In the H. family, the second child died of the infantile type of the disease under one year of age and the third child is still alive at the age of ten years, having had visual symptoms since the age of eight but is normal mentally and has a cherry-red spot at the macula. Sjögren, in his study of fifty-nine cases of the juvenile type could find no example of the infantile type in his families. *It has been impossible after an exhaustive search of the literature to find any example of a family in which infantile and juvenile types of the disease have occurred.*

5. *The disease may run a chronic course.*—Several examples

of cases which have survived considerably longer than the age of two years are on record. In Peterson's family one child lived to the age of five and a half years, and Koller mentions a patient alive, but an idiot, at the age of four years. In the Ha. family, the third child (Case 10) was well, apart from visual symptoms at the age of ten years, and in the L. family two children are alive but idiots at the age of seventeen and seven years respectively. Case 7 had its first symptoms at the age of ten months but did not die until almost four years of age.

6. *The presence of a cherry-red spot in some members of a family with only optic atrophy in others.*—Such an association was present in Higier's (1906) family, and in our L. family.

7. *Congenital cases may occur.*—Norman and Wood have recorded a case of amaurotic idiocy dying at the age of eighteen days with typical histological findings but did not record any retinal changes.

B. JUVENILE TYPE.

1. *Occurrence in Jews.*—Occasional examples of this have been recorded as in the family cited by Higier (1896) and that of Kuffler.

2. *The presence of a cherry-red spot in the retina.*—No example of this has been recorded. In Wilson's book there is a drawing of a fundus with a cherry-red spot, stated to be present in a man of twenty-five with no other symptoms. This unfortunately was an error and the fundus drawing was that of Case 7 (aged 3½ years). Our Case 10, while occurring in this age group was obviously of the infantile type suggesting strongly therefore that *two types of the disease occur which are not identical.*

3. Families have been recorded in which the typical macular changes are present in some members while optic atrophy only is present in others, *e.g.*, Kuffler, and Torrance. It would seem probable that optic atrophy may precede macular changes in some cases, as in the infantile type.

4. *Juvenile cases occurring in the late infantile period.*—Families are recorded in which the onset of symptoms may be in the typical juvenile period in some members but in the third to fifth years or late infantile period in others. For example, F. E. Batten's typical juvenile cases occurred in the third year, Ichikawa's in the fifth year and Torrance's in the second year. In Mühlberger's family one child aged three and a half years had macular changes resembling the juvenile type, while the other child of fifteen months had a normal fundus. Bielschowsky's four cases, all showing optic atrophy only, occurred in the fourth year. In Vogt's second family one child showed the first signs of the disease at four years and died three years later, the second began to have

failure of vision at four years, dying two years later, while the third began to fail in the second year and died within the third year. Case 11 began to have symptoms at the age of three years and had typical eye changes at the age of four and a half years.

5. Although the patients usually die by the age of eighteen years they occasionally survive much longer as in our Cases 21, 23 and 24.

6. One of our cases (9) was born with a tumour on the ear.

C. LATE INFANTILE TYPE.

Under the title " Late Infantile Familial Amaurotic Idiocy with Cerebellar Symptoms " Bielschowsky described an intermediate group between the infantile and juvenile types of the disease. His cases began in the fourth year and the eye signs appeared late and usually consisted of optic atrophy only. Histologically the cerebral lesions are the same as those in the two other types. It is usually considered that cases with their onset between the ages of one and four years fall into this group. Although attempts have been made to differentiate this type of the disease from the two preceding, it is doubtful if such is possible. We have seen above that the cherry-red spot may occur in this period and that a patient exhibiting this sign may be one of a sibship in which the onset of the condition is usually in the infantile period. Further, in some families in which some members have a typical cherry-red spot, others have merely optic atrophy and either of these findings may be present, whether the case occurs in the infantile or late infantile periods. We have also noted that families exist in which some members of a sibship have the typical macular changes of the juvenile type and others only optic atrophy. *No family has been described in which a cherry-red spot has occurred in some members and the peppered pigmentary changes in others.* Optic atrophy is a common sign of degeneration of the retina and it may occur in association with a cherry-red spot or the pigmentary retinal changes of juvenile cases, or alone. The last finding has been suggested by some authors, *e.g.*, Greenfield and Nevin, as the basis of considering such cases occurring in the late infantile period as transitional between the two main groups, but, as we have seen, it may occur in association with either of the two main types. There are no grounds to justify the separation of a late infantile type from the infantile and juvenile types. In the period from one to four years may occur cases of the infantile type having an onset later than usual and also, cases of the juvenile type coming on earlier. All that can be said is that, when cases occur in this period the retinal changes tend to be those of optic atrophy only, but both types of the disease may occur.

D. LATE JUVENILE TYPE.

Apart from the two main types previously considered occasional cases with typical retinal changes and histological findings may occur with the first symptoms showing themselves at puberty or shortly afterwards. Such cases have been mentioned by Kufs, and by Paton, but little attention has been paid to them previously. Two further families of this type are now described, the Da. and

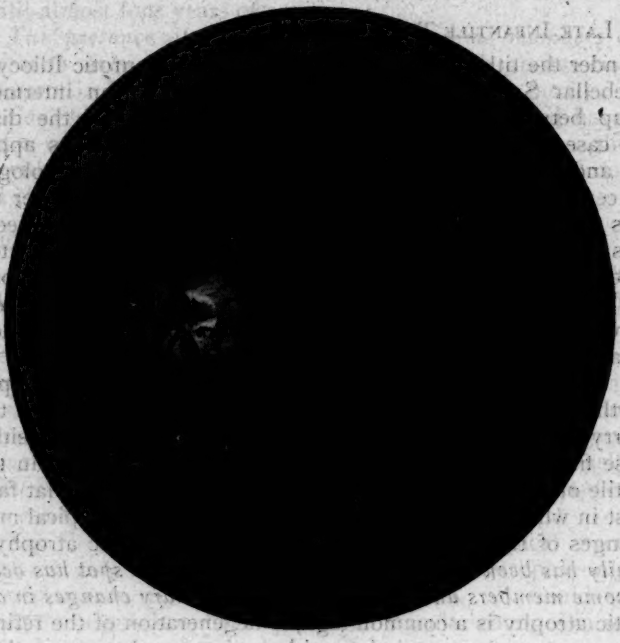


FIG. 9.

Fundus of a late juvenile case. (Case 26.)

Ho. families, Cases 25-27. They resemble the juvenile cases in the retinal changes except that these tend to be less marked than usual (see Fig. 9). Only in one of our cases did any convulsions occur and then only a small number at the beginning of the illness. It is obvious that these cases are the same as the juvenile type, but with a later onset, as in Case 27 there was a younger brother who died from the disease at the age of six years. The course tends to be slower and the symptoms of the disease chiefly due to cerebellar and pyramidal tract involvement causing considerable progressive ataxia, spasticity and dementia.

The Ho. Family

Parents alive and well, English, from Halifax, Yorks. Two children, a boy (Case 25) and a girl (Case 26). No nervous disease on either side of the family. The mother had two brothers and a sister but all had died. Grandparents healthy. The father had two sisters alive and well.

CASE 25. Joseph Ho.

History.—Full-time child, instrumental delivery, breast-fed for 4½ months, "milestones" normal. At the age of twelve he had a few convulsions but these only lasted for a week and there had been none since then. At the age of fourteen he began to drag one leg. He was able to walk, however, until the age of eighteen when both legs had become very stiff and his arms began to shake so much that he spilt things and was unable to write. His speech became affected. Later he became childish, but not facile. Towards the end his sight began to fail and he became completely bedridden for the last two months of his life and quite helpless. There was no sphincter disturbance. He died at the age of twenty-six years of cerebro-macular degeneration.

CASE 26. Kathleen Ho., aged 24 years. Admitted to the National Hospital on November 21, 1928, under the care of Dr. Collier.

History.—Normal delivery. Breast-fed to the age of 13 months. "Milestones" normal. Went to school at the age of six and considered to be quite normal. She left school at the age of fourteen and was considered to be normal or perhaps a little backward mentally. She went to work until the age of eighteen. At the age of sixteen she was noticed to be stooping a little and this had tended to get worse. At the age of seventeen both legs began to get stiff and this interfered with her working and caused her to give up a year later. Since that time the stiffness had gradually become more marked, causing an aching in the legs, which frequently let her down and caused her to fall. In the last three years she had used her hands very little for knitting or writing but there had been no tremor. Two years before admission she began

to laugh excessively and increasingly so, and about the same time complained of failure of vision but was still able to read on admission. Throughout her illness there had been some hesitancy in commencing micturition. Her speech had also grown somewhat slower but there had never been any convulsions or loss of weight.

Examination.—Gross degree of dementia. Visual acuity 6/24 right and left. Fundi: (Mr. Paton), vessels rather numerous, scattered patches of pigment in perimacular region with a little redness round each. Small area of atrophy close to macula with a little irregular pigment, discs being normal. Both eyes similar (Fig. 9). Fields: relative central scotoma in both eyes. Very slight tremor of outstretched hands with intention tremor on movement. Spastic paraplegia. No ataxia in lower limbs. Sensation normal. All tendon jerks brisk. Abdominal reflexes only just elicited. Bilateral extensor plantar responses. Gait, spastic right more than left. Hands and legs red and cold with some oedema below the knee. Nil palpable in abdomen. C.S.F. normal. Pr. 0·04 per cent.

The patient remained unchanged while in hospital.

The Da. Family

Father alive and well from the London area. Mother alive suffering from left hemiparesis of nine months duration. The children were:—

1. Case 27, a boy.
2. A younger brother suffering from convulsions and blindness and he died at the age of six years from cerebro-macular degeneration.
3. Stanley, aged thirteen, had a fit aged three weeks but none since. He wore glasses at the age of five. At the age of nine he had an operation on his left eye for squint. Examination showed visual acuity on right to be 6/9 and left had movements only. Fundi normal.

CASE 27. Dennis Da., aged 20 years. Admitted to the National Hospital, December 17, 1934, under the care of Dr. Martin.

History.—From birth to the age of fifteen years he was abnormally fat, at the latter age weighing twelve stone eight pounds. Seven months before admission, while playing tennis, he found difficulty in following a fast moving ball and in judging its distance from him. Three months later he had difficulty in seeing in the dark. He could see car lights but not people, especially if they were standing still, and, a month later still, he noticed tiny moving spots dancing in front of his eyes. One month before admission, at night the sky seemed to be pink, but the moon and stars appeared yellowish-white. During the past two years he had

noticed a shaking of his hands when he was nervous and, if he placed his toes on the ground when sitting, they went into clonic movements.

Examination.—A well-built man, six feet in height. Quiet, intelligent and co-operative. Not emotional. Memory and speech normal. Visual acuity 6/36 right and 6/60 left. Fundi: (Mr. Paton) at the maculae of both eyes was an area about half a disc in diameter consisting of a pigmented network around a hyperaemic area suggesting early cerebro-macular degeneration. Discs normal. Pupils, large, react normally. No other physical signs in the central nervous system. Spleen and liver not palpable. C.S.F. normal. Pr. 0·05 gms. per cent.

E. ADULT TYPE.

Apart from the cases considered, a few have been described with typical histological findings occurring in adults. When this happens it is common to find that the whole syndrome is not present, either the retinal or cerebral symptoms being absent.

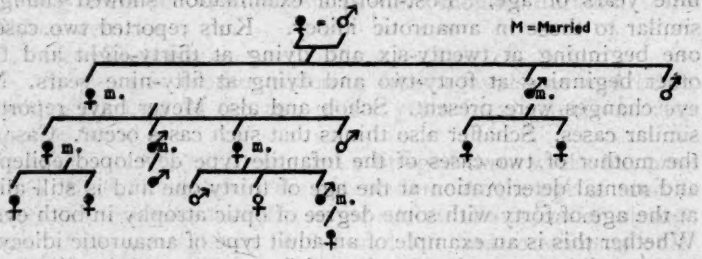
1. *Adult amaurotic idiocy without eye signs.*—Walter reported on a family of which three members were affected with idiocy and nervous lesions but showed no eye changes. The youngest died at twenty-three but the others were alive at twenty-four and twenty-nine years of age. Post-mortem examination showed changes similar to those in amaurotic idiocy. Kufs reported two cases, one beginning at twenty-six and dying at thirty-eight and the other beginning at forty-two and dying at fifty-nine years. No eye changes were present. Schob and also Meyer have reported similar cases. Schaffer also thinks that such cases occur. Case 8, the mother of two cases of the infantile type developed epilepsy and mental deterioration at the age of thirty-one and is still alive at the age of forty with some degree of optic atrophy in both eyes. Whether this is an example of an adult type of amaurotic idiocy it is impossible to say, but in view of the presence of the disease in two children it would seem probable that such was the case. It must be a matter for conjecture as to whether two types of the disease exist comparable to the two types of amaurotic idiocy.

2. *Eye signs occurring alone.*—Oatman suggested such cases occurred and he has recently been supported by Kufs, who thinks that some late juvenile cases do not develop eye changes, but it is common to find that one or more ascendants of such a case have typical retinal lesions but never develop any mental or motor symptoms. The father of Ford's case had *pigmentary degeneration* of the retina from early life with only slow deterioration of vision. In Case 24 it was only by careful observation that any mental or physical changes could be noticed and apart from the

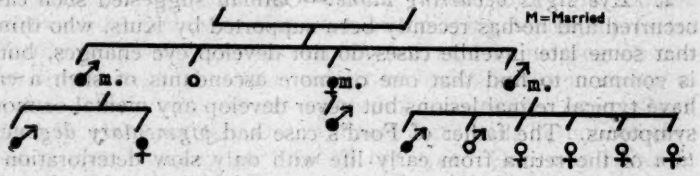
retinal changes there were no abnormal physical signs. No cases in which macular changes of the infantile type (cherry-red spot) occurred alone have been reported previously but such was present in Case 10.

The condition of macular heredo-degeneration is brought to mind by the above cases. This may occur at different ages ranging from an infantile to a senile type; most cases have their onset between six and twenty years. The evidence that these cases are related to amaurotic idiocy is doubtful. Clinically the macular changes are similar but the fact that no families have been reported in which the two diseases have occurred together and the presence of pigmentary degeneration of the retina in the father of Ford's juvenile case would seem evidence that the diseases are not associated. This contention receives support when the mode of inheritance of macular heredo-degeneration is studied. It appears to be inherited in a dominant manner. Examples of two such family trees are given below.

(*a*) The Re. family, living in London, a member of which was seen at the Royal Westminster Ophthalmic Hospital on January 17, 1940, by Mr. J. G. Milner. The onset of symptoms in this family occurred about the age of puberty and the pigmentation was present as fine flecks at both maculae.



(*b*) The Pa. family, with onset of symptoms about the age of seven or eight years. This family also lived in London and one member was seen at the Royal Westminster Ophthalmic Hospital recently.



It seems improbable therefore that heredo-degeneration is related to amaurotic idiocy.

The Relationship of Amaurotic Idiocy to the Lipoidoses

In 1916, Knox, Wahl and Schmeisser observed the occurrence of Tay-Sachs' disease (infantile) in cases of Niemann-Pick's disease and this had since then been noted on a number of occasions. Lindau, in 1930, found the typical changes in the nervous system in a case of Gaucher's disease and Ashby et al. case of Gargoylism. Earlier histologists had stressed the fact that although the cells in Tay-Sachs' disease may be filled with pre-lipoid, the fibres of the cells may be normal and argued that its presence does not represent the products of nerve cell degeneration but a faulty general metabolism with fat deposition in the nervous tissues. In Niemann-Pick's disease there is a widespread lipoid histiocytosis with deposition of phosphatides (lecithin) most prominently in the spleen and liver, but also in other organs such as bone-marrow, adrenals, lymph nodes and blood. The blood cholesterol may be raised to 650 mgms. per cent. but may be normal. The serum may be cloudy and there may be large vacuoles of lipoid material in the lymphocytes, polymorphs and monocytes, and this may be diagnostic. The similarity in the reaction of the abnormal cell contents in the liver in Niemann-Pick's disease and in the brain in Tay-Sachs' disease is very striking. Tay-Sachs' disease has been regarded by Bielschowsky, Kufs, Spielmeyer, etc., as a localised histological expression of a metabolic disorder. On the contrary, Schaffer and others have thought that only the Niemann-Pick's disease was the result of metabolic disorder and that Tay-Sachs' disease was due to a lipoid degeneration of the nervous tissue, the fat originating locally, and Rintelen supported this view on the ground that in a case of Niemann-Pick's disease, with a cherry-red spot at the macula, the optic nerve was histologically normal. Van Bogaerts observation, in which Tay-Sachs' and Niemann-Pick's diseases occurred in the same family, is, however, strong evidence of unity in the pathology of these affections, and our observation in Case 2 of a raised blood cholesterol seems to furnish evidence that a generalised metabolic disturbance is present. Evidence of unsuspected lipoid changes in the liver, spleen, etc., in amaurotic idiocy has been present in some infantile cases but not in the juvenile type, thus Brouwer found " foam " cells in the spleen of an infantile case. Similar evidence was found by Pick, Schob and Walter. Norman and Wood in their congenital case found involvement of the reticulo-endothelial system in a minor degree in the spleen, liver, suprarenals and lymph nodes, in which lipoid deposits were present in the cells. The histological finding in the brain varied from the normal in the histochemical

reactions of the intracellular lipoid and the deposition of extracellular cholesterol crystals. In this regard the findings of an enlarged liver and spleen in Case 6 and of raised blood lipoids in Case 10 are significant, whereas in Case 2 the faecal fat content and splitting were normal.

Klenk has recently shown that in juvenile amaurotic idiocy there is present in the brain an unknown lipoid substance with a melting point of 195°C. and containing nitrogen and sugar. In this respect it resembled the other lipoidoses such as Gaucher's disease in which abnormally high amounts of cerebrosides are present in the liver and spleen, and Niemann-Pick's disease in which sphingomyelin infiltrates the parenchyma of the various organs. From a histochemical point of view, the lipoid in the cells in amaurotic idiocy is to be regarded as composed of chemical substances allied to those entering into the formation of normal myelin. From its solubility it is probably related to the normal cerebrosides. There is reason to believe that in the lipoidoses some step in the metabolic processes of lipoids is faulty resulting in the storage of unmetabolised products and that this is due to lack of specific enzymes differing in the different types of diseases and in the different types of amaurotic idiocy.

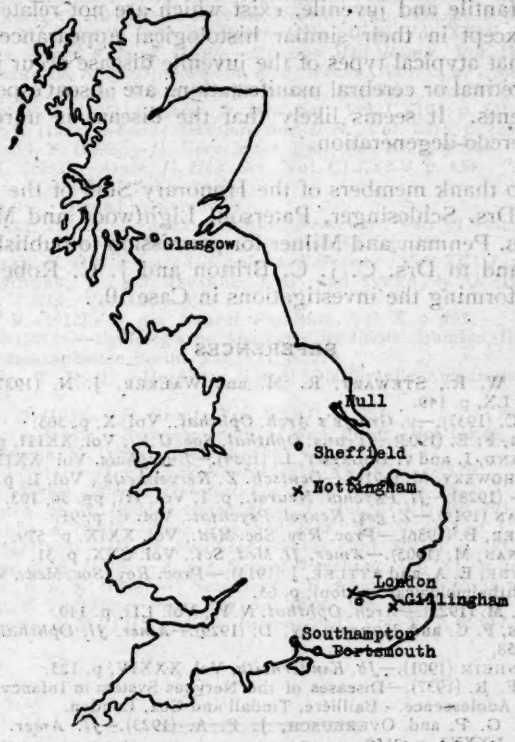
Case 10 deserves further consideration. On the unitary conception of all forms of amaurotic idiocy a case occurring at the age of eight years would be expected to show the retinal changes usually present in the juvenile type of case, but in Case 10, on the contrary, a cherry-red spot was found, no mental or neurological symptoms being present. On the supposition that Tay-Sachs' disease is part of a general lipoid dystrophy, of which Niemann-Pick's disease is the full syndrome, this case can be regarded as a monosymptomatic form of such a lipoidosis. By extensive investigation evidence of general lipoid disturbance could be obtained in the high blood fats. Behr has described late forms of the Christian-Schuller syndrome of a monosymptomatic type, no change in the blood lipoids being found. This disease was previously regarded as infantile and Case 10 may be looked on as a congruent form in Niemann-Pick's disease.

With the above facts in mind, attention must be drawn to the histochemical findings. In the infantile type of the disease the substance deposited in the cells of the nervous system and retina is of a " pre-lipoid " nature, whereas in the juvenile type it is of a lipoid nature, the two types differing in this respect. Again, as already mentioned, no record of the association of Niemann-Pick's disease with the juvenile type of amaurotic idiocy has occurred either directly or in a family. Niemann-Pick's disease, like the infantile type of the disease, is especially liable to affect Jewish

children. It is probable therefore that two distinct types of amaurotic idiocy exist:—

1. An infantile, mainly confined to Jews, usually, but not always, rapidly fatal and almost always with the onset before the third year. Occasionally only optic atrophy occurs instead of the usual macular cherry-red spot. It is probably related to Niemann-Pick's disease.

2. A juvenile, with onset chiefly about six to eight years of age, but sometimes as early as the second year or as late as the twenties. It occurs chiefly in non-Jews, has a slower course and also occasionally shows optic atrophy instead of the pigmentary macular changes, the final retinal picture resembling retinitis



X = Cases of infantile type reported in England and Scotland.
O = Cases of juvenile type reported in England and Scotland.

Geographical distribution of cases of infantile and juvenile amaurotic idiocy in this country.

pigmentosa. It is unrelated to the infantile type genetically or histochemically and its relationship to macular heredo-degeneration is doubtful. Often only cerebral or retinal changes are present in older cases.

Geographical Distribution of Cases

The map shows the geographical distribution in England and Scotland of my own and other reported cases.

Summary

A number of typical and atypical cases of amaurotic idiocy are reported and evidence is brought forward that two types of the disease, infantile and juvenile, exist which are not related to one another, except in their similar histological appearances. It is probable that atypical types of the juvenile disease occur in which either the retinal or cerebral manifestations are absent especially in older patients. It seems likely that the disease is unrelated to macular heredo-degeneration.

I wish to thank members of the Honorary Staff of the National Hospital, Drs. Schlesinger, Paterson, Lightwood and Moncrieff, and Messrs. Penman and Milner for permission to publish records of cases, and to Drs. C. J. C. Britton and J. C. Robertson for kindly performing the investigations in Case 10.

REFERENCES

1. ASHBY, W. R., STEWART, R. M. and WALKER, J. N. (1937).—*Brain*, Vol. LX, p. 149.
2. BEHR, C. (1937).—*v. Graefe's Arch. Ophthal.*, Vol. X, p. 303.
3. BATTEN, F. E. (1903).—*Trans. Ophthal. Soc. U.K.*, Vol. XXIII, p. 386.
4. BERTRAND, I. and v. BOGAERT, L. (1934).—*Encéphale*. Vol. XXIX, p. 505.
5. BIELSCHOWSKY, M. (1914).—*Deutsch. Z. Nervenheilk.*, Vol. L, p. 7.
6. ——— (1928).—*Jl. Psychol. Neurol.*, p. 1, Vol. III, pp. 36, 103.
7. BRODMAN (1914).—*Z. ges. Neurol. Psychiat.*, Vol. C, p. 91.
8. BROUWER, B. (1936).—*Proc. Roy. Soc. Med.*, Vol. XXIX, p. 579.
9. BUCHANAN, M. (1905).—*Amer. Jl. Med. Sci.*, Vol. CXX, p. 31.
10. COCKAYNE, E. A. and ATTLEE, J. (1914).—*Proc. Roy. Soc. Med.*, Vol. V, pt. 3 (Ophthalmological Section), p. 65.
11. COHEN, M. (1923).—*Arch. Ophthal. N.Y.*, Vol. LII, p. 140.
12. CONDES, F. C. and HORNER, W. D. (1929).—*Amer. Jl. Ophthal.*, Vol. LII, p. 558.
13. FALKENHEIM (1901).—*Jb. Kinderheilk*, Vol. XXXIV, p. 123.
14. FORD, F. K. (1937).—Diseases of the Nervous System in Infancy, Childhood and Adolescence. Bailliére, Tindall and Cox, London.
15. FRETS, G. P. and OVERBUSCH, J. F. A. (1923).—*Jl. Amer. Med. Ass.*, Vol. LXXXI, p. 2158.
16. GIL, R. and BERANGER, R. P. (1930).—*Arch. Oftal. B. Aires.*, Vol. V, p. 36.
17. GORDON A. (1907).—*N.Y. Med. Jl.*, Vol. LXXXV, p. 295.
18. GREENFIELD, J. G. and HOLMES G. (1925).—*Brain*, Vol. XLVII, p. 183.
19. GREENFIELD, J. G. and NEVIN, S. (1933).—*Trans. Ophthal. Soc. U.K.*, Vol. LIII, p. 170.

20. HASSIN, G. B. (1926).—*Arch. Neurol. Psychiat. Chicago*, Vol. XI, p. 708.
21. HASSIN, G. B. and PARMELEE, A. H. (1928).—*Amer. Jl. Dis. Child.*, Vol. XXXV, p. 87.
22. HIGIER, H. (1897).—*Deutsch. Z. Nervenheilk.*, Vol. IX, p. 1.
23. ——— (1901).—*Neurol. Zbl.*, Vol. XVIII, p. 843.
24. ——— (1906).—*Deutsch. Z. Nervenheilk.*, Vol. XXXI, p. 231.
25. ICHIKAWA K. (1909).—*Klin. Mbl. Augenheilk.*, Vol. XLVII, p. 73.
26. KLENK, E. (1939).—*Hoppe-Seyl. Z.*, Vol. CCLXII, p. 128.
27. KNOX, J., WAHL, R. and SCHMEISSER, H. C. (1916).—*Johns Hopk. Hosp. Bull.*, Vol. XXVII, p. 1.
28. KOLLER, C. (1896).—*Trans. Amer. Ophthal. Soc.*, Vol. VII, p. 661.
29. KUFFLER, O. (1910).—*Beitr. Augenheilk.*, Vol. VIII, p. 18.
30. KUFS, H. (1928).—*Z. ges. Neurol. Psychiat.*, Vol. CXVII, p. 753.
31. ——— (1929).—*Z. ges. Neurol. Psychiat.*, Vol. CXXII, p. 395.
32. LIENER, J. H. and GOODHART, S. P. (1927).—*Arch. Neurol. Psychiat. Chicago*, Vol. XVII, p. 616.
33. LEVY, A. (1923).—*Amer. Jl. Ophthal.*, Vol. VI, p. 408.
34. LINDAU, A. (1930).—*Acta Psychiat.*, Vol. V p. 167.
35. MAGNUS, H. (1885).—*Klin. Mbl. Augenheilk.*, Vol. XXIII, p. 42.
36. MARINESCO, G. (1930).—*Jl. Psychol. Neurol.*, Vol. VIII, pt. 1, pp. 41, 1.
37. MAYOU, M. S. (1904).—*Trans. Ophthal. Soc. U.K.*, Vol. XXIV, p. 142.
38. MUGGIA, A. and VITA, A. (1930).—*Minerva Med. Roma.*, Vol. II, p. 5.
39. MUHLBERGER, A. (1903).—*Münch. Med. Wschr.*, Vol. L, p. 1,968.
40. NORMAN, R. N. and WOOD, M.—*Jl. Neurol. Psychiat.*, Vol. IV (N.S.), p. 175.
41. OATMAN, E. L. (1911).—*Amer. Jl. Med. Sci.*, Vol. CXLII, p. 221.
42. PATON, L. (1932).—*Trans. Ophthal. Soc. U.K.*, Vol. LIII, p. 226.
43. PETERSON, F. (1898).—*Jl. Nerv. Ment. Dis.*, Vol. XXV, p. 529.
44. PICK, L. (1932).—*Amer. Jl. Med. Sci.*, Vol. CLXXXV, p. 453.
45. POOLEY. Quoted by Falkenheim.
46. RINTELEN. Quoted by Sorsby.
47. ROGALSKI, T. (1910).—*Arch. Psychiat. Nervenkr.*, Vol. XLVII, p. 1,195.
48. SACHS, B. (1887).—*Jl. Nerv. Men. Dis.*, Vol. XIV, p. 541.
49. SCHAFFER, K. (1931).—*Arch. Psychiat. Nervenkr.*, Vol. XCIII, p. 767.
50. SCHLESINGER, B., GREENFIELD, J. G. and STERN, R. O. (1934).—*Arch. Dis. Child.*, Vol. IX, p. 1.
51. SCHOB, F. (1912).—*Z. ges. Neurol. Psychiat.*, Vol. X, p. 303.
52. ——— (1912).—Pathologische Anatomie der Idiotic Bumkes, Handbuch der Geisteskranheiten Berlin, Vol. VII, p. 779.
53. SJÖGREN, T. (1931).—Hereditas (Lund) (Die juvenile Amaurotische Idiocy), p. 14.
54. SLOME, D. (1933).—*Jl. Genet.*, Vol. XXVII, p. 363.
55. SORSBY, A. (1937).—*Brit. Encyclop. of Med. Pract.*, Vol. III, p. 30.
56. SPIELMEYER, M. (1906).—*Neurol. Zbl.*, Vol. XXV, p. 57.
57. ——— (1918).—*Jl Psychol. Neurol.*, Vol. XXXVIII, p. 120.
58. TAY, W. (1881).—*Trans. Ophthal. Soc. U.K.*, Vol. I, p. 55.
59. TORRENCE, H. W. (1922).—*Glasg. Med. Jl.*, Vol. XCVII, pp. 193, 1.
60. TURNER, J. A. (1912).—*Proc. Roy. Soc. Med.*, Vol. V, pt. 1, p. 3.
61. VOGT, H. (1905).—*Mschr. Psychiat. Neurol.*, Vol. XVIII, p. 161.
62. WALTER, F. K. (1918).—*Z. ges. Neurol. Psychiat.*, Vol XL, p. 349.
63. WILSON, S. A. K. (1940).—"Neurology," E. Arnold, London, ii, p. 882.
64. WEBER, F. P. (1910).—*Proc. Roy. Soc. Med.*, Vol. III, p. 58.
65. WOLFSOHN, J. M. (1915).—*Arch. Intern. Med.*, Vol. XVI, p. 257.

MARGINAL HAEMORRHAGE ON THE DISC. PARTIAL CROSS TEARING OF THE OPTIC NERVE. CLINICAL AND HISTOLOGICAL FINDINGS

BY

PROFESSOR ARNOLD LOEWENSTEIN

TENNENT INSTITUTE OF OPHTHALMOLOGY (PROFESSOR W. J. B. RIDDELL)

GLASGOW

BLUNT injuries of the eye lead to different ophthalmoscopic pictures. Retinal and choroidal haemorrhages combined with blood in the vitreous body are common, as are choroidal rupture and retinal detachment. Rupture of the posterior ciliary arteries, evulsion of the optic nerve and bleeding within the optic sheath are found less frequently. The latter occurs in the frequent cases of fracture of the base of the skull and is frequently combined with papilloedema. But even very gross skull fractures do sometimes occur with insignificant changes at the disc.

H. Knapp (1868), according to Wagenmann, described a case with red optic disc and a ring-shaped haemorrhage. A short note by Bunge (1905) mentions a case of basal skull fracture with papilloedema, haemorrhages within the disc and surrounding it. The disc became pale and a red half-ring remained. Bunge assumed that a bleeding of the optic sheaths occurred within the sclera.

Describing an ophthalmoscopic picture which is not mentioned in the literature as far as we are aware, we put forward an explanation on the basis of some seemingly new histological experiences.

Cl. C., aged 38 years, was injured, while at work, by a long nail falling upon his right eye from a considerable height. His vision was blurred. When he was admitted to the clinic on September 12, 1942, a perforating wound was found in the sclera at " 4 o'clock," 2 mm. from the limbus. The cornea was clear, anterior chamber deep, 2 mm. hyphaema, pupil small, not reacting, lens clear, a dull reflex from behind without showing any fundus details, severe hypotonia. Vision right = fingers at ½ m. Vision left = 6/6. X-ray negative. Atropine did not dilate the pupil more than 5 x 4 mm. Fourteen days later, besides dark diffuse vitreous opacities, a clear red arcuate band was visible at the temporal margin of the disc from " 11 to 7 o'clock " (Fig. 1). The breadth was approximately that of the central vein, with both ends slightly thinned. The shining red arc was outlined temporally by delicate specks of the pigment ring. On October 7, 1942, the picture was much clearer, as the blood in the vitreous had mostly

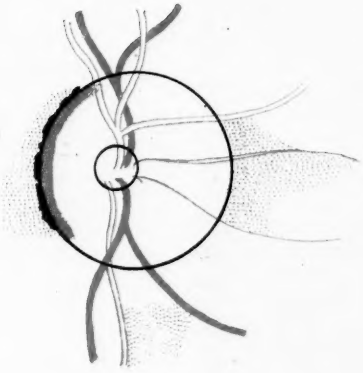


FIG. 1.

Arcuate haemorrhage at the disc with radial retinal bleeding.

disappeared. The marginal haemorrhage was unchanged and remained so during the next two months of observation. Three fine radially striate haemorrhages, which seemed more superficial, were present temporal, nasal and inferior to the disc (Fig. 1). The inferior one was the last to be discovered (October 13, 1942). The radial haemorrhages were persistent too, but were becoming fainter on November 13, 1942, and had vanished entirely by November 27, 1942, while the arcuate marginal haemorrhage remained unchanged. The vitreous body was much clearer, and some floating opacities were visible in the lower half.

The field of vision in the left eye was full with normal blind spot, while the field of the right eye showed slight concentric shrinking, more in the nasal part. I was unable to determine the blind spot of the injured eye. The question of the Jensen scotoma remains unsolved, unfortunately, due to a lack of collaboration on the part of the patient.

As we had not the opportunity of making a histological examination in this case, we are forced to analyse this picture by speculation only, drawing our conclusions from other histological findings. The first and nearest conjecture is that the red crescent is caused by a bleeding within the optic sheaths, subdural or subarachnoidal—infiltrating Elschnig's border tissue. As is well known, the latter separates sclera and choroid from nerve tissue. The pigment epithelium often contains less pigment near the optic nerve, or ceases entirely. In this case, such a border tissue haemorrhage would shine through the pellucid lamina vitrea choroideae, and the marginal haemorrhage would be outlined temporally by the pigment ring. We cannot offer any histological proof for this assumption.

Ballantyne (1942) has shown that in cases of intracranial subarachnoid haemorrhage, subdural and subarachnoidal haemorrhages in the sheaths of the optic nerve, as well as those in the retina, occur as independent effusions. He has found nothing to prove that a subarachnoid haemorrhage may track forward from the blind end of the optic vagina into the border tissue or surrounding sclera. He found once only (his Case 1) rows of red blood corpuscles within the nerve fibre and ganglion cell layer close to the disc tracking from the border tissue into the retina. This case was observed by Ballantyne ante mortem ophthalmoscopically. Haemorrhages crossed the lower temporal margin of the optic disc. No arcuate haemorrhage was visible.

It seems necessary to remind ourselves that neither retina, choroid nor sclera are in direct touch with the tissue of the optic nerve. Choroid and sclera are separated from the nerve by Elschnig's border tissue, while the retina ends a short distance from the nerve. This distance is filled with Kuhnt's intermediary

tissue. *The only membrane of the ocular tissue reaching the optic nerve directly is the lamina vitrea choroideae* (Bruch). It butts closely against the optic nerve.

Studying the histological material of injured eyeballs collected in the Tennent Institute, I did not discover a case of bleeding in the border tissue. But I found a remarkable lesion of the optic nerve in three cases—all three at the very place where Bruch's membrane terminates at the optic nerve.

Case 1

Do. P., aged 40 years. While drunk he was attacked from behind and struck with a knife or some instrument, which hit his left eye. Rupture of the sclera in lower outer quadrant with prolapse of uvea and retina. Anterior chamber full of blood. Tension = —2, amaurosis. Excision of the eye.

Histological examination :—Subepithelial granulation mass containing uvea, vitreous body, polynuclear leucocytes, erythrocytes and haemorrhagic retina. Iris ruptured at its root. No lens. Retina totally detached, covered on both sides with blood, its vessels well filled. The optic nerve is torn temporally at the level of the hexagonal layer. The gap runs transversely in general. The tear is step-shaped, notched (Figs. 2 and 3). The steps are at intervals, whose breadth is that of an optic nerve bundle group. The gap is filled with blood. A smaller tear is found at the nasal side. Between the ends of both cross tears is normal nervous tissue.

FIG. 2.

Case 1. Tear crossing the nerve fibres temporally. Low power.

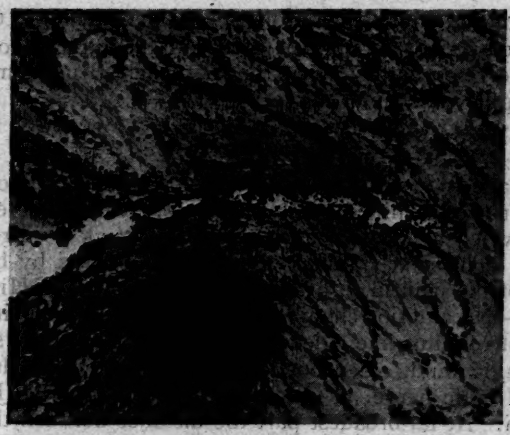


FIG. 3.

Case 1. High power. Notched cross tear, gap filled with blood. The rupture is the direct continuation of the pigmented epithelium.

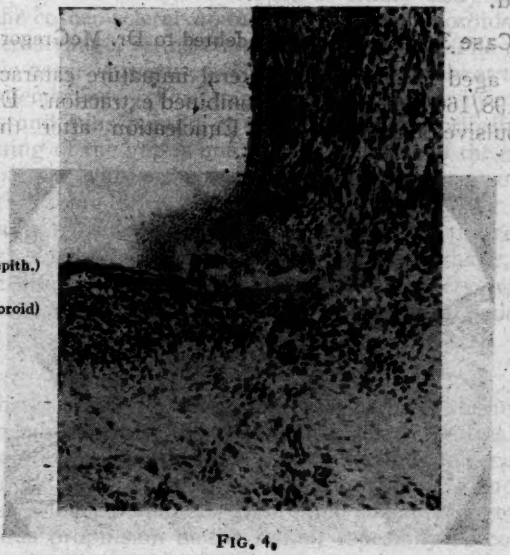


FIG. 4.

Haem. Eos. 150×. Haemorrhagic glaucoma with complete retinal detachment. Partial tear in direct continuation of Bruch's membrane. Gap filled entirely with blood.

A perforating injury with prolapse of the contents of the globe and complete retinal detachment caused an incomplete cross tear of the optic nerve on both sides at the spot where Bruch's membrane encloses tightly the optic nerve.

Case 2

F. M., aged 62 years. Bilateral glaucoma discovered in 1931. Left eye trephined, 1931, right eye, 1936. Both eyes quiet till 1939, when a haemorrhagic glaucoma attack occurred in the right eye. The next (January, 1940) was followed by blindness. Excision of the painful right eye, February 3, 1940. Histological findings :—No anterior chamber, uvea entirely cellular infiltrated, retina, convolvulus-like, detached by an extensive subretinal haemorrhagic fluid. Leucocytic and lymphocytic infiltration of retina and optic nerve. Cross-tear of the optic nerve (Fig. 4) of 90μ length. At its broadest part the distance between the side of the gap is 45μ. The whole gap is filled with blood. The tear is the direct continuation of Bruch's membrane.

A haemorrhagic glaucoma with endophthalmitis and haemorrhagic retinal detachment revealed a short tear of the optic nerve at the very insertion of Bruch's membrane. The gap is filled with blood.

Case 3 (for which I am indebted to Dr. McGregor)

C. M., aged 79 years. Bilateral immature cataract. Blood pressure 198/160 mm. Left eye combined extraction. Eight hours later expulsive haemorrhage. Enucleation after three days.

FIG. 5.

Case 3. Partial cross rupture in expulsive haemorrhage. Low power.

FIG. 6.

Case 3. Partial cross rupture in expulsive haemorrhage.

Anatomical examination:—Prolapse of ciliary body with retinal tissue in the corneo-scleral wound, extensive subchoroidal and subretinal haemorrhage, retina completely detached. The nasal third of the optic nerve is ruptured (Figs. 5 and 6) exactly at the level of Bruch's membrane. The gap is partly filled by red blood corpuscles and runs parallel to the fibres of the cribriform plate. The outlining of the gap is notched. The tissue of the optic nerve in front of and within the lamina cribrosa is cavernous. Both central vessels are filled with blood.

An expulsive haemorrhage led to prolapse of the contents of the globe, subchoroidal and subretinal haemorrhage, complete retinal detachment. The nasal side of the optic nerve was torn to the extent of about ½ mm. exactly at the ending of Bruch's membrane.

These three cases of a tear in the optic nerve at the insertion of Bruch's membrane were discovered in a relatively small number of eyes examined histologically. They happened to occur in three different conditions: (1) trauma, (2) haemorrhagic glaucoma, and (3) expulsive haemorrhage. Subretinal haemorrhage with an instantaneous propulsion of the retina, especially as corollary of arterial bleeding, seems to be the common cause. In all three cases the tear occured exactly at the insertion of Bruch's membrane. Our Case 2 revealed a tear and haemorrhage of about 90μ length

at the periphery of the nerve. If that happens in an eye with translucent media and retina in situ we can expect an ophthalmoscopic picture similar to that which we have put at the head of this paper. Such a haemorrhage may give rise to an intraretinal bleeding, too. The striate character of the latter is explained by the thick radial nerve fibre bundles at the optic disc margin. The retinal haemorrhage is thinner than the blood column in the rupture, therefore more quickly reabsorbed. It is a pity that the search for a Jensen scotoma, corresponding to the tear in the nerve fibre, was frustrated by the lack of co-operation of the patient.

Cross ruptures of the optic nerve have been reported by von Michel (1901) in the area of the cribriform plate in two cases. Both cases were perforating injuries. In the first, the whole nerve was cut at the anterior level of the cribriform plate. The second case showed two incomplete tears on opposite sides, both filled with blood—like our Case 1.

It seems that the insertion of Bruch's membrane is a point where tears of the optic nerve may occur more easily than anywhere else. Not only a traction from the detached retina may produce a tear. If the nerve is pushed backwards by a stronger force—for example, a foreign body such as a bullet—the nerve fibres may rupture at the same place. Actually we know that appearance as evulsio nervi optici clinically and anatomically. Salzmann (1903) described it, and many such cases have been published. Good ophthalmoscopic pictures are available in Lagrange's and in von Szily's books on war injuries, and in Würdemann's text book. The often considerable space between the retracted optic nerve and the retina is filled in fresh cases with blood, substituted later by glial and connective tissue. I remember eight cases, notes of four of them being at my disposal. Two are injuries by bullets, two birth traumata. The youngest (9-year-old boy) was the only one with a very deep "cup." Two were cases with intensive proliferant retinitis, one with optic atrophy. In all these cases the mechanism of the rupture is understandable. The force behind the bulb pulls the nerve backwards and (or) pushes the bulb forwards. The rupture of the optic nerve occurs at the locus minoris resistentiae, the insertion of the nerve at the retina. If the acting force is not strong enough to cause an evulsion of the optic nerve, complete or incomplete tears occur, as in von Michel's two cases, in front of the cribriform plate. The tear in our three cases was caused by an instantaneous subretinal haemorrhage.

Summary

A seemingly unknown ophthalmoscopic picture, marginal arcuate haemorrhage at the periphery of the disc, is described. It is explained as a tear of peripheral nerve fibres at the insertion

of Bruch's membrane at the optic nerve. Three cases of partial cross ruptures of the optic nerve are reported histologically. All three are discovered at the very insertion of the lamina elastica chorioideae. The tear was caused in all cases (traumatic, expulsive haemorrhage and haemorrhagic glaucoma) by an instantaneous, probably arterial, subretinal bleeding. Traumatic retropulsion of the optic nerve or propulsion of the bulb causes cross ruptures of the optic nerve, too, occurring at the same spot and leading to evulsion of the optic nerve.

REFERENCES

BALLANTYNE.—Mackenzie Memorial Lecture: Vascular Disease and its Significance in Medical Ophthalmology. *Glas. Med. Jl., Vol. XX, p. 26, 1942.*

LAGRANGE, F.—Atlas d'Ophtal. de Guerre, Masson, Paris, 1918.

VON MICHEL.—*Zeitschr. f. Augenheilk.*, Vol. VI, p. 1, 1901.

SALZMANN.—*Zeitschr. f. Augenheilk.*, Vol. IX, p. 489, 1903.

VON SZILY.—Atlas der Kriegsaugenheilkunde. F. Enke, Stuttgart, 1918.

WAGENMANN.—Verletzungen des Auges. Graefe Saemisch-Handbuch der Augenheilkunde, 3rd Ed., II, Chap. 17, 1915.

WÜRDEMANN, H. V.—*Injuries of the Eye.* London, 2nd Ed., 1932.

ORTHOPTIC TREATMENT OF ANOMALOUS PROJECTION

BY

DIANA S. MANN, B.Sc.

MELBOURNE

WHILE theory concerning the nature of abnormal retinal correspondence or anomalous projection has been considerably developed in the last ten years, notably by the writings of Chavasse, Verhoeff and Travers, practice in its treatment has hardly kept pace. In this paper an attempt is made to outline briefly the theoretical conceptions which prove to be of most practical value in guiding an extension of training methods. Of the many names under which the phenomenon goes, Verhoeff's term, "anomalous projection," is adopted as being most in accordance with these conceptions.

Ordinary binocular vision, in which both visual axes are always directed on the same object by reflex fusion mechanism, is an innate habit, for although it develops after birth, it does so along predisposed lines. The same applies to the mental processes that interpret the two images received, one by each macula, as being a single object (if the images are similar), or as representing two

objects in the same place (if the two images are dissimilar). A further innate habit is suppression of one macular image when they are dissimilar, as often happens when a distant object is hidden from one eye by a nearer object; while one eye is fixed on the far object, the image of the nearer falls on the macula of the other eye, but in most cases it is only by careful attention that one can see it. In squint the faculty of suppression may be developed simply by constant use, and it being part of the innate visual mechanism, this is done without difficulty.

On the other hand, those who react to squint by developing what is known as abnormal correspondence or anomalous projection, do so by maintaining a constantly high level of attention on the two unlike macular scenes so as to keep one separated from the other, and to project each approximately to its true position. Anomalous projection requires a high level of attention because it is not an innate tendency, but is an abnormal use of the visual nervous mechanism; it may become habitual, but imposes a greater nervous strain than the normal reflex of suppression. Suppression of one macular field in squint is developed by concentrating attention on the other conflicting field. A direct means of attacking suppression, therefore, is to excite attention in the previously neglected direction. An equally direct means of attacking anomalous projection is harder to find, because divided attention between two conflicting macular fields is probably influential in its genesis. One finds that in anomalous projection of recent origin, a pair of unlike pictures, such as used for testing S.M.P. on the synoptophore, will be perceived as widely separated when placed one in front of each eye, while a fusible pair of pictures may be not only fused, but seen stereoscopically.

Practising such fusion may be used as the first step in the recovery of normal vision. It cannot, it is important to remember, be used to straighten the eyes in a convergent squint at this stage since, if abduction is attempted, the eyes fail to maintain fusion and can be seen to move as the patient looks from one picture to the other, though he will insist that he sees only one. Presumably, by producing homonymous diplopia, one has imitated the conditions under which anomalous projection was developed, so that the patient's mind reverts to this state. However, the response to attempted adduction is far better and, in convergent squints, one may develop some range of fusion (between the resting angle and a more convergent one) by this means; the patient is aware of diplopia when convergence fails. Even in divergent squints, if of the type where ability to converge on near objects is not lost, the patient may maintain genuine fusion by convergence. He will not be aware of diplopia when fusion breaks, but will notice loss of stereoscopic depth there.

The possibility of fusion and stereopsis without normal S.M.P.* is important in regard to the treatment of anomalous projection, but two other facts are equally so. One is that the disruption of normal correspondence does not apply over the whole binocular field. This is shown by the apparent behaviour of images when synoptophore tubes are moved from zero towards the angle of squint. If one eye, let us say the right, be fixed on a lion, the image of the cage will fall on an eccentric part of the left retina, which normally receives the duplicate (which is disregarded) of what the right eye is fixing. Once attention is aroused to this cage, it is perceived as lying to the left of the lion; and if attention and fixation are firmly centred on the lion, and the cage is moved steadily towards the angle of squint, the cage is seen to move towards the lion and (in a fairly recent anomalous projection) momentarily surrounds it before it "jumps" away to the right. The apparent behaviour previous to the jump is exactly that which would occur under similar conditions in normal projection, and is as if the cage image fell on the normally corresponding points in the eye that is fixed on the lion. The jump occurs when the cage image falls on a part of the left retina, *i.e.*, the macula, where the attention level is sufficiently high for it to be recognised as a left retinal image, and therefore to be projected to the right side of the lion. The jump occurs sooner in an old-established anomalous projection, where a high attention level has spread to a greater distance from the macula; and when steady monocular fixation is not maintained it occurs much earlier still—usually when the cage image reaches a point about 15° from the angle of (convergent) squint, for the disappearance of the non-fixed image as it falls on the blind spot induces an immediate movement to fix it with the macula, which projects it accurately with respect to the image seen a moment before with the other macula.

When the tubes are moved from the angle of squint towards zero, the "jump" back occurs at a point nearer zero than the "jump" in the opposite direction, as once the individual projection of each retina is excited, normal projection reasserts itself with more difficulty.

The third point is that the apparent "abnormal correspondence" of the macula of one eye with a point some 15° from the macula of the other eye hardly exists until it has been aroused by tests and measurements. A frequent description of the pictures as seen with two eyes is that they seem to be "in different worlds," and the indecision of the patient whose prolonged efforts to "put the lion in the cage" may ultimately be abandoned as impossible;

* S.M.P. is here used for the sake of brevity in the generally accepted sense, as implying normal macular correspondence in the presence of unlike macular images as well as simultaneous perception.

or by fixing on a position where "I can't see them both properly, but it must be in the cage somewhere about here," indicates that he is governed by no direct sense of their relative projection, but rather is trying to reconcile two conflicting impressions. The apparent illogical failure of his own senses inevitably excites the interest of the patient, whatever his age, and if allowed he will make repeated attempts to cage the lion by movements from either side, and to convince himself that in some position they coincide. Not only does this help to settle the abnormal relationship more firmly in his mind, but it encourages an analytical attentive mental attitude; whereas normal correspondence is best regained by relaxing mental vigilance to give the innate nervous mechanism a chance. It is, on the whole, unwise even to measure the angle of anomaly, as this may start a false train of thought in the patient's mind.

Treatment of Anomalous Projection—PRE-OPERATIVE.

1. Maintain constant total occlusion of one eye except during orthoptic exercise, so that the need for independent projection disappears.

2. Do not measure the angle of anomaly except in special cases, when one should indicate by one's manner that the results are unimportant.

3. During treatment the patient must watch the pictures constantly, but for most exercises will do best if his attention is distracted by stories, conversation about home affairs, discussion of some detail in the appearance of the pictures, or any subject other than the relative position of the two pictures before him. To maintain the right state of attentive inattention is the most difficult and most important part of the orthoptist's work. Each patient will require different methods of approach, and will respond best to different combinations of the methods outlined in 4 and 5.

4. Normal correspondence may be encouraged most readily by inserting fusion pictures, preferably with fusion check and some stereoscopic element at the angle of squint, locking the tubes and (1) shaking them in front of the patient in a quick jerky manner, (2) rapidly alternating the lights behind the pictures, (3) making the patient shut his eyes for a moment and then look again. Once a single image is perceived and held, attention to check marks and stereopsis is encouraged. When fusion can be maintained in the primary position, adduction (in convergent squints) or abduction (divergents) should be developed, to encourage fusion reflex movements without risk of falling back on abnormal correspondence.

5. Normal correspondence is not firmly established until S.M.P. slides are seen together at the true angle of squint. Methods of achieving this are:—

(*a*) The patient fixes the lion while the cage is swept up steadily from outside the angle. It is important that fixation should be steady, the story being that "the lion will get frightened if he knows the cage is coming, so we must pretend not to know it's there." The cage tube should be carried on a little past the angle of squint, and then moved backwards and forwards for a while across it, as long as the cage seems to be passing across the lion. The subsequent return movement of the tube to the outward position must be done quickly while talking so that the reverse approach of the cage is disregarded. If the patient takes an interest in the return movement, or if he fails to maintain steady fixation on the lion, this exercise must be abandoned.

(*b*) The patient maintains fixation on the lion, while the cage tube is moved backwards and forwards across the angle.

(*c*) The tubes are locked together at the angle and shaken.

(*d*) If the patient is intelligent, one may carry on a modified form of "chasing." First it is demonstrated to him that at the true angle the pictures are both seen at once more clearly than in any other position (although they appear to be separated in space). The orthoptist then moves one of the tubes, and the patient moves the other so as to bring the pictures again to the clearest position. This exercise must, of course, be abandoned if the patient attempts to approximate the pictures at the "false" angle.

In all of the above one may, of course, use any other pair of S.M.P. slides, using very large angle ones at first, and finally the smallest.

6. When all traces of anomalous projection with S.M.P. pictures has disappeared, and some range of duction has been developed as in (3), reduction of the squint by abduction (convergent squints) or adduction (divergent) may be attempted. Constant vigilance, however, is necessary to make sure that the patient is not suppressing and returning to anomalous projection.

7. Operation is almost inevitably necessary, and there is no point in prolonging treatment before operation if the patient shows no improvement in three or four visits. The only general prerequisites are that the vision should be equal and the patient should be able to alternate readily. When time is short, as with country patients, operation should be performed as soon as this state is attained. The choice of operation will be affected by the following considerations:—

(*a*) Operative reduction or correction of the squint inevitably leads, in time, to reduction of the angle of anomaly, and often restores normal correspondence surprisingly quickly, or speeds up the response to orthoptic methods.

(*b*) A good operative result (angle 0 to —5°) after convergent squint obviates the necessity for developing S.M.P., as this is not required as a pre-requisite for adductions, but may develop automatically as the fusion range increases. Adduction improves rapidly with convergence exercises on near objects, as fusion, however weak, is then reinforced by accommodation-convergence.

(*c*) A post-operative convergence after convergent squint, on the other hand, is difficult to treat unless good S.M.P. and fusion amplitude are present. If there is any tendency to suppression, this will increase rapidly to counteract diplopia in the new position.

(*d*) Certain cases, much quoted in text-books but fortunately rare, tend to recur after operation. This may possibly be due to annoyance caused by inaccurate post-operative projection and diplopia, but may happen even when anomalous projection has disappeared. Recurrence is most likely to take place when the angle before operation is variable with a considerable accommodative element, and the patient is of a nervous and excitable type. Although it would appear that a special effort should be made to obtain normal correspondence before operation in such cases, they are usually particularly stubborn ones; the long training may have the effect of concentrating attention on the eyes, so that any nervous excitement acts as a direct stimulus to convergence, and the squint may recur in spite of recovery of normal correspondence. The question is complicated and is probably one in which use of sedatives and attention to psychic and home influence is of more importance than orthoptics.

8. *Post-Operative.*—When the operative result is good, the probability is that some binocular vision may be recovered in time without orthoptic treatment. However, the recovery is seldom complete, and the patient may develop typical eye-strain symptoms at once or years afterwards which clear up when a good fusion range is developed. If symptoms do not develop or are not combated until some years after operation, habits of suppression may by this time have grown too strong to be overcome, and one is unable to relieve the symptoms. It is therefore best to give orthoptic treatment until a range of fusion of at least +25° to 0° with some stereopsis is obtained.

Treatment is essentially the same as pre-operative, making allowance for the altered angle of squint with consequent change in position of the "false correspondence" angle. Exceptions are in post-operative divergence after convergent squint. First, occlusion is contra-indicated for such cases unless there is very

marked suppression and divergence. Secondly, as there is no need to increase power of abduction, it is unnecessary to insist on S.M.P., though this is, of course, an advantage. The power of convergence may be greatly developed by practice on a pencil, etc., as for convergence deficiency, and one often finds that S.M.P. is attained readily after such a stimulus to normal binocular vision.

Should the abnormal correspondence not yield readily to treatment after operation, the angle being satisfactory, it is probably wisest to leave the case alone, with observation at intervals, so that when time has done its bit in breaking down the abnormal correspondence, one may jump in and deal the death blow.

Although the treatment as here indicated includes some time-honoured methods, the omission of several useful tricks will be obvious to orthoptists. My aim has been to make a statement as simple as possible.

CONTACT LENS IN CASES OF NEUROPARALYTIC KERATITIS

BY

M. KLEIN

LONDON

AMONG the indications for the use of contact lenses neuroparalytic keratitis does not seem to have attracted much attention. This record of their use in two cases may therefore be of interest.

1. Miss V. P., aged 39 years. Had several operations for right sided acoustic tumour. The last operation left her with a complete right facial palsy and trigeminal anaesthesia. Neuroparalytic keratitis of the right eye developed, which eventually made tarsorraphy necessary, but in spite of this, the state of the cornea did not improve. After a year, though the lids were still sutured, the condition of the eye seemed hopeless. The therapeutic effect of a contact lens was then tried as a last resort (August, 1942). Rapid improvement followed, and within 2–3 weeks the eye became white, the cornea cleared up considerably, except at the central area where a dense scar remained. The patient is wearing the contact lens regularly from 7 a.m. to 1 p.m. when it is removed for about half-an-hour, and generally worn again till about 9 p.m.

An incident emphasised the value of the contact lens. In November, 1942, the patient accidentally broke the contact lens. The eye became inflamed within a few hours, and by the following

From the Central London Ophthalmic Hospital.

day the corneal infiltrate was extensive. The development of such a severe keratitis in such a short time was astonishing. The patient was then supplied with a new contact lens, and within a week the eye quietened down again.

2. Mrs. A.D., aged 58 years. Five weeks after alcohol injection for right trigeminal neuralgia, neuroparalytic keratitis developed. When first seen a fortnight after the onset of the affection the area involved extended centrally for about 7mm. diameter. The patient was supplied with a contact lens, which she is wearing for about 12 hours a day, with a break of about half to one hour at mid-day. The patient has now been wearing the contact lens for four months. The cornea has cleared considerably, though there is a central nebula. Vision without contact glass but with correction is 6/24, and with contact glass 6/12.

Comment:

1. *Fitting.*—From the fitting point of view the anaesthesia of the eye is a slight initial advantage. The taking of the impression can be made without the use of any anaesthetic. When the contact lens is put in, the reaction of the eye makes it easy to tell whether the fitting of the contact lens is satisfactory. A wrong fitting causes irritation (hyperaemia, discharge, etc.). In the absence of subjective signs in the neuroparalytic cornea the reaction of the eye shows readily any deficiency of the fitting.

2. *Indications.*—It appears that the use of contact lenses is warranted as an alternative to tarsorraphy. It gives better protection for the eye as was shown in the first case. From the aesthetic point of view it has distinct advantages. In the first case the patient was much happier with the contact lens which has enabled her to discard the shade she previously wore to hide the deformity caused by the sutured lids. Moreover, there need be no reluctance in trying a contact lens when there is hesitation about undertaking such a disfiguring procedure as tarsorraphy.

My thanks are due to Miss J. M. Fleming for referring the first case to me, and Miss A. Logan Adam for the second case.

ORBITAL EMPHYSEMA SIMULATING CELLULITIS

BY

C. L. CONOR O'MALLEY

GALWAY

ORBITAL emphysema is apparently a rare condition as indicated by the literature at my disposal on the subject. The majority of cases are described as following a benign course, not necessitating surgical

interference. I have found no description of a case similar to the following:—

Description of Case

Patient Michael W., an agricultural worker was looking on at a football match, from a position behind the line. A player charged over, collided with him, and stuck his finger or fingers into patient's left eye. There was slight haemorrhage from the eye and the left nostril. The eye was closed but there was no pain on that, or the following day. On the third day after injury, while at work in the beet-field, about the noontide hour, he felt his eye "beginning to bulge." The condition became more acute towards evening and was accompanied by severe pain. That same night he saw his doctor, who ordered him to hospital, where he arrived next day. I saw him shortly after admission, *i.e.*, on the fourth day after injury. Here is a note of his symptoms, general and local:—

Patient was very ill, in a state of partial collapse, and suffering intense pain. The temperature was 99·5° and pulse 100. Notwithstanding the low temperature, the general condition suggested an acute infection. There was marked proptosis of the left eye, which protruded between the lids, leaving a central belt of cornea completely exposed. The lids were stretched, tense like a drum. Fleshy folds of chemosed conjunctiva overhung the cornea, and projected over the edges of the lids. The eyeball was stony hard from external pressure. The cornea was opaque, and had sloughed at the area of exposure, causing a large irregular ulcer. No detail of intra-ocular structures could be seen, and vision was reduced to nil. The eyeball was immobile, and the orbital tissue felt hard and brawny. There was no trace of a wound from the intruding finger, and examination of the nose was negative.

Treatment and Course

On admission patient had warm saline irrigations of the conjunctiva, atropine, and an injection of M. and B. 693 soluble. The cornea was threatened both from exposure necrosis, and by strangulation from the tense eyelids, and swollen conjunctiva. Orbital cellulitis was the diagnosis, and without delay, under a general anaesthetic, an incision was made along the middle third of the upper margin of the orbit. When a sinus forceps was pushed through behind the eyeball, a quantity of gas bubbled up through the blood in the wound. The eyeball immediately subsided into the orbit in a most dramatic fashion, the cornea was safely covered by the lids, and the tension in all the orbital structures was forthwith relieved. The orbital incision was kept open for three days, and was then allowed to close permanently. Although no pus, or foul smell was manifest, and gas infection of the orbit is said to be a very rare occurrence, yet

this possibility could not be immediately excluded.[1] Cultures from the depths of the wound, however, proved negative, except for an apparently accidental contamination with staphylococcus albus. There was a slow but steady recovery. For quite some days the cornea seemed to hang in the balance; but at the end of a week the ulcerated area showed signs of healing, and the periphery began to clear. Eventually the cornea cleared throughout, with the exception of a nebula just below the pupillary area. As the cornea cleared, two small posterior synechiae could be seen, and also a retinal haemorrhage near the disc; otherwise the intra-ocular structures were normal. Chemosis took 28 days to resolve. The severity of the chemosis was possibly due to an infection by dirty fingers as much as by obstruction of the circulation due to intra-orbital tension.

Ptosis was very persistent, so much so, that one feared a lesion of the levator or its nerve twig; at the end of six weeks, however, ptosis had disappeared, and diplopia which had come on cleared up a few weeks later. Ultimately the patient recovered 6/9th vision with full field and movements. The retinal haemorrhage vanished, and except for adhesions and a small nebula the eye became normal.

Discussion

Although repeated radiographs failed to show a break in the sinus walls bounding the orbit, this was obviously a case of orbital emphysema, but with symptoms indicative of cellulitis. Radiography of the thin sinus walls offers many technical difficulties, and probably it is not possible to demonstrate a hole in the lamina papyracea. The sequence of events leading to the exophthalmos was not typical of emphysema cases. A finger pushed into the orbit had presumably fractured the lamina papyracea, and opened a way into the ethmoid. It is unlikely that the slight trauma of a finger would fracture into the frontal sinus or antrum. The patient could not associate the onset of proptosis on the third day with the blowing of his nose or sneezing; but this must have been the immediate cause of his emphysema. There was no evidence of intra nasal disease.

McArthur[2] reviewed 33 cases of facial emphysema. In 10 cases there was evidence of pre-existing bone disease, leading to fracture on slight trauma. Davis[3] states that the majority of cases of emphysema give a history of sudden increased pressure in the buccal and nasal cavities. Van Duyse[4] describes a case of orbito-palpebral emphysema following a fracture of the skull, which involved the orbital wall of the frontal sinus. The emphysema disappeared and recurred over a period of time. Terrien[5] classified emphysema about the orbit as: orbital, orbito-palpebral and palpebral. Caccialupi[6] described in detail a case of fracture of the infero-internal angle of the orbit, involving the frontal and ethmoid sinus,

and causing orbito-palpebral emphysema. His case is illustrated by radiographs, which demonstrate very clearly the pathway of air into the orbital tissues. The majority of cases of emphysema, described in the literature, seem to have been caused by violent blows with obvious fracturing of the sinus walls adjoining the orbit. Fuchs[7], however, says that orbital emphysema may be produced by blows on the eyeball, pushing it backwards against the orbital fat. The orbital fat is thereby displaced against the orbital wall, with sufficient violence to fracture the lamina papyracea, thus opening a pathway for air to enter the orbit. If the eyeball is ruptured by the blow the recoil on the orbital fat is neutralized, and so fracture of the lamina with emphysema does not occur. Therefore according to this author the presence of one lesion excludes the other. It is difficult to visualize the eyeball resisting a blow sufficiently violent to fracture indirectly the orbital wall. Würdemann[8] enumerates three symptoms which he thinks are characteristic of orbital emphysema. (1) Exophthalmos lessened by deep inhalation. (2) The eyeball may be pushed back into place with a feeling of crepitus, and a sound like crunching snow. (3) The eye protrudes on blowing the nose. He apparently assumes a free to and fro passage of air from sinus to orbit. This type of injury would presumably not cause dangerous pressure on, or exposure of the eye.

Most writers on the subject think that the various forms of orbital emphysema can be adequately treated by a pressure bandage and by cautioning the patient against sneezing and blowing his nose. It is clear, however, that cases can occur such as that herein described, where strangulation of the eyeball is threatened, and to relieve the tension, an immediate evacuation of the air is imperative.

Summary

A case is described of a severe proptosis which endangered the eye, and was caused by a gaseous accumulation in the orbit. The symptoms were indistinguishable from those seen in orbital cellulitis. An evacuation operation to relieve the proptosis became urgent. The eye which looked a total loss, made an almost complete recovery.

By inference one concludes that here was a case of orbital emphysema. The air may have got behind the patient's eyeball, directly through a stab wound made by the football player's finger. This is unlikely. The more likely explanation is, that the finger made a valve like opening in the orbital plate of the ethmoid which allowed air under pressure to enter the orbit, but not to leave it. The case seems unusual in that the eye was in great danger; whereas orbital emphysema is usually not a serious condition, and requires no treatment other than a pressure bandage over the eye.

REFERENCES

1. MCGREGOR, I. S.—*Brit. Med. Jl.*, p. 292, February 28, 1942.
2. MCARTHUR, L. L.—*Jl. Amer. Med. Assoc.*, Vol. XLIV, p. 1418, 1905.
3. DAVIS, G. G.—*Sur. Gynec. and Obstet.*, Vol. XXIV, pp. 761-762, 1922.
4. VAN DUSYE, M.—*Bull. Soc. Belg. Ophtal.*, Vol. LXXIII, pp. 61-64, 1936.
5. TERRIEN, F.—*Soc. Franc. Ophtal. Traite Ophtal.*, Vol. VI, pp. 345-346, 1939.
6. CACCIALUPI, P.—*Saggi Oftal.*, Vol. III, pp. 331-337, 1927.
7. FUCHS, E.—Diseases of the Eye.
8. WÜRDEMAN.—Injuries of the Eye.

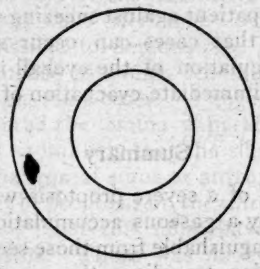
REMOVAL OF SMALL MAGNETIC FOREIGN BODY FROM THE EYE EIGHTEEN MONTHS AFTER THE DATE OF INJURY*

BY

HUMPHREY NEAME

LONDON

HENRY B., aged 34 years.—The patient attended Moorfields Eye Hospital on July 6, 1940, complaining of soreness of the left eye, the result of the entry of powdered chalk into it nine days previously. He gave a history of having injured the left eye 18 months before



Site of slight depression on surface of left iris, with pigmented floor and faint diffusion of rusty colour around on the iris stroma.

while knocking off the head of a screw with a hammer and chisel. The eye had been slightly red at the nasal side ever since.

On examination the left eye showed marked ciliary injection. There was a corneal scar to the nasal side and a dark rust coloured nodule on the iris behind the scar (see figure). The corneal microscope and slit-lamp revealed marked aqueous flare, a few very fine deposits on Descemet's membrane, and small round dark spots, evidently distributed in the posterior reflecting zone. (These spots

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were considerably smaller than the ordinary colloid body (Henle-Hassel body), and have on several occasions been seen as a transient manifestation in inflamed eyes).

On July 6, 1940, by means of the Haab magnet the foreign body was freed from the iris and it was removed through a keratome section. The patient was discharged from Hospital on July 13, 1940, with the eye in a quiet condition.

On July 20, 1940, some fine deposit was visible on the back of the cornea with the microscope. The posterior corneal reflecting zone showed good endothelial pattern with a few of the dark spots already mentioned. There was no pigment or deposit on the anterior surface of the lens. After retinoscopy R.V. with +0·5 D.Sph. +0·5 D.Cyl., axis 90°=6/6. L.V. with +1·25 D.Cyl., axis 30°=6/9.

On August 10, 1940, the left eye was completely white. There was a slight depression noticeable on the iris, with a pigmented floor and slight diffusion of rusty brown colour in the immediate neighbourhood only.

The weight of the fragment of metal was found to be 0·0025 gm.

It seemed to the writer worth reporting this case, as an indication of the fact that very minute magnetic foreign bodies may be retained without setting up any appreciable amount of siderosis during a period of considerable length.

A CASE OF MULTIPLE INTRA-OCULAR FOREIGN BODIES

BY

R. A. D. CRAWFORD

LONDON

ON November 25, a man, aged 40 years, was stamping out steel aeroplane parts on a hand press when he felt a sting in his right eye. His eye was bathed by the first aid nurse, but in the evening he found the vision blurred, and saw a black shadow across his right field.

He was admitted to Moorfields next day in the care of Mr. Maurice Whiting, when the condition of the right eye was:—white eye; a small perforating wound above centre of cornea; the anterior chamber and iris normal; a track of a foreign body through the lens; in the vitreous—"A," a fine glistening foreign body about 3 mm. long, lying antero-posteriorly, with one end in the centre of the disc; "B," a smaller similar foreign body at "6 o'clock" at the bottom of the vitreous; two minute foreign bodies in the vitreous near the macula; small dusty vitreous opacities; the retina showed slight

oedema around the disc; the tension was normal. Vision 6/36. The left eye was normal and read 6/5.

"A" was found to be weakly magnetic when the Haab magnet was used under direct ophthalmoscopic observation.

The magnet was used once or twice a day from November 26 till December 9, when "A" came forward clear of the disc; this was accompanied by a small haemorrhage temporal to the disc which cleared entirely in three days.

Two days later "B" came forward behind the iris at 7 "o'clock," and remained here until brought into the anterior chamber and removed by hand magnet through a keratome section on December 18.

Meanwhile the excursions of "A" became from day to day somewhat greater, but it was held by a strand in the vitreous, and returned always to the same position, where it was focused with a +12 lens in the ophthalmoscope.

After a respite of ten days the giant magnet was again used on December 28, and on the 29th "A" came into the anterior chamber. It returned through the pupil almost as soon as the magnet was switched off, but was removed through a keratome section by the hand and giant magnets.

The patient was discharged on January 3, 1943. The right eye was then white, the cornea showed two keratome incisions, one above and one on the temporal side, and a small scar above its centre from the perforating wound. There was no K.P., the anterior chamber was normal, the pupil under atropine, the lens clear without a trace of a track on ophthalmoscopic or slit-lamp examination. The vitreous showed some general haze and fine strands. A small darkened area in the centre of the optic disc marked the place where "A" had lain, and there was some pigment disturbance below at the site of "B." The minute foreign bodies seen on admission in front of the macula were not now seen. The tension was slightly below that of the left eye (which read 6/5 and was normal). Investigation on the Bjerrum screen showed a normal blind spot and no scotoma.

Right Vision was 6/18, improved to 6/12 by $\frac{+0{\cdot}50 \text{ D.Sph.}}{+0.50 \text{ D.Cyl., } 90°}$

The points of interest in the case would appear to be the entrance of multiple weakly magnetic foreign bodies, the lodging of one in the optic disc, and the removal of the first after three weeks and the second after five weeks treatment and about 28 sessions with the Haab giant magnet, in spite of the disappointing result at first obtained.

These factors, especially the involvement of the optic disc, made the prospect of employing the posterior route particularly uninviting. So weakly magnetic were the particles that an attempt to remove them by scleral incision and hand magnet might easily have failed and trauma been inflicted to no purpose.

That the particle "A" could so easily be observed while under the giant magnet made the generally increasing response easy to assess; but the result was the same in the case of "B," which could not be observed and yet was successfully removed.

One must conclude that however poor the initial response, it is unwise to abandon the giant magnet for small magnetic intra-ocular foreign bodies until a very prolonged trial has been made. Ophthalmoscopic observation during application of the magnet is of great assistance, as the excursion obtained may increase gradually although the resting position may remain the same for a long period. Even if observation is impossible, persistence may succeed, though it is much more difficult to decide whether progress is being made.

It is not claimed that the final result in this case will be a first class eye, but it is suggested that less damage was inflicted than would have resulted from employment of the posterior route.

The patient has been seen two weeks after his discharge; the vision of the left eye had improved to 6/9 with correction.

I wish to thank Mr. Maurice Whiting, F.R.C.S., and Surgeon Rear Admiral H. Whiteside, R.N., for their permission to report this case.

ANNOTATION

Insidious Cases of Glaucoma

If one thinks of the number of cataract extractions performed each year one must admit that the sequel of raised intra-ocular pressure is not very common. But it does occur sometimes and the sort of case we have in mind would be an admirable test for a candidate in an examination for a higher diploma in ophthalmology, one which, if properly investigated, would give ample opportunity for the examinee to prove his worth. For such cases are not simple cataracts or simple glaucomas; they partake of each and the signs, particularly those on the glaucomatous side, are apt to be anomalous. When one sees a patient with a highly congested eye and steamy cornea that can be seen from a distance it is usually a fairly simple matter. But with a quiet eye and clear cornea the presence of raised tension is not so obvious and the mind may easily get side-tracked into looking for other reasons for defective vision, such as lens capsule, macular change and what not. We call to mind a case of this sort which came to us from a remote part of the country

which we should certainly have missed had not the local Aesculapius, in a covering letter, said that on one of his visits he thought that the eye was harder than it should have been. The patient had undergone cataract extraction, and had been subsequently needled at least three times. He was really suffering from secondary glaucoma. When we saw him he had very little field left. It is not easy to be wise at all times and in all places. How often when patients make an appointment does one hear them say, "I won't keep you a moment"? If one takes their momentary statement at its face value one may find when he arrives that one is in for at least half-an-hour's work before he can be got rid of. With the need for keeping other appointments there is a real danger that some part of the routine examination may be scamped. Such a thing happened once to a surgeon of our acquaintance. The patient was in a hurry and was fitted in at the last moment. He said his sight had "gone funny" in the train and wanted to be assured that there was nothing wrong. The surgeon found nothing amiss, though the examination of the fundus was not easy, as the patient was very intolerant of bright light. A week or two later he had a recurrence of his symptoms. When asked how his sight had gone funny, he answered, "I saw a coloured ring round a light in the carriage on my way home." Had our friend only persisted in enquiries as to what the patient had observed, he would never have missed the chronic glaucoma present.

ABSTRACTS

I.—THERAPEUTICS AND OPERATIONS

(1) **Mckee, S. Hanford (Montreal).—Gonorrhoeal ophthalmia neonatorum treated with sulphanilamide.** *Canadian Med. Assoc. Jl.*, August, 1939.

(1) **McKee** reports a case of ophthalmia neonatorum in an infant two weeks old. The disease had started on the second or third day following birth. Each eye was affected with profuse discharge and films made from it showed the Gram-negative coffee-bean shaped diplococci. The child was admitted to hospital and in addition to the usual local treatment was given daily 2 gr. of sulphanilamide every four hours in 5 doses. The weight of the infant was 8lb. Rapid improvement took place; on the fourth day the discharge had practically ceased and the infant was discharged

at the end of a week. Films made on the third day showed no micro-organisms.

The author also refers to a case of adult gonorrhoeal ophthalmia which did very well on this treatment and was published in the *Arch. of Ophthal.*, 1939, Vol. XXI, p. 1035.

R. R. J.

(2) **Ballantyne, A. J. (Glasgow)—Problems of vision in aviation.** *Glasgow Med. Jl.*, March, 1940.

(2) A very interesting post-graduate lecture on problems of vision in aviation was delivered in the Tennent Memorial Institute by **Ballantyne** on September 5, 1939, and is reprinted here.

The author deals with visual standards for both civilian and military air pilots, emphasises the paramount importance of a full field of vision, goes into the questions of depth perception, and the one-eyed flyer. He discusses in some detail the physiological disturbances to which a pilot is exposed both with regard to the ocular apparatus and the disturbances of circulation of the blood. The paper is too technical to yield readily to a short abstract and should be read in the original. Two diagrams by Wing-Commander Livingston are reproduced to show the "black out" produced by turning sharply at the bottom of a dive and that effected by a "tight loop."

R. R. J.

(3) **Abramson, A. W. and Flacks, B. (Leicester).—Staphylococcal septicaemia complicating carbuncle of the face. Recoveries after treatment with M. and B. 693.** *Lancet*, Vol. CXXXVII, p. 1065, 1939.

(3) **Abramson** and **Flack's** paper will interest ophthalmic surgeons for its account of the treatment which prevented cavernous sinus thrombosis in 2 out of 6 patients, in 5 of whom staphylococcal septicaemia was proved.

Two patients recovered from staphylococcal septicaemia following in one case a boil on the right side of the upper lip and in the other on the right cheek. The early use of M. and B. 693 in large doses, ligature of the angular vein under local anaesthesia and the intravenous administration of 2,500 units of anti-staphylococcal serum were of value. In one case where the patient was vomiting M. and B. 693 was given intra-venously 3 c.c. diluted with 10 c.c. of distilled water for 4 injections and then continued 4 hourly intramuscularly for a night and a day until vomiting had ceased and it was possible to continue with oral administration of 1·0 grm. 4 hourly.

H. B. STALLARD.

II.—CONJUNCTIVA

Turtz, C. A. (New York).—Verruca as a cause of unilateral conjunctivitis. *Amer. Jl. Ophthal.*, Vol. XXV, p. 452, 1942.

Turtz describes two cases of unilateral conjunctivitis of long duration which resisted all forms of local treatment but were eventually cured by the removal of minute warts near the lid margin, hidden by the eyelashes. He discusses the infectious nature of warts, their production by a filterable virus and their auto-inoculable property.

H. B. STALLARD.

III.—LENS

(1) **Clapp, C. A. (Baltimore, Maryland).—Alterations in the capsular epithelium in immature cataracts.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 437, 1942.

(1) Clapp has made a histological study of 70 cases of immature cataract removed in the capsule. Atrophy of the capsular epithelium was found in practically all the cases. The epithelial cells were flattened, in some instances absent over wide areas, and in many cases the nuclei had migrated. Proliferation of these cells was evident in a few cases. This paper is illustrated by 14 microphotographs, 3 of which show normal capsular epithelium at 25, 59 and 74 years respectively. The remainder are from pathological material. The author discusses the rôle of the capsular epithelium in relation to the biochemistry of the lens, its permeability and its part in pathological processes. He suggests that alterations in and death of the capsular epithelium are a cause rather than a result of lens changes.

H. B. STALLARD.

(2) **McLean, J. M. (New York).—Cataract extraction after glaucoma operation.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 192, 1942.

(2) McLean describes a modified technique for cataract extraction in a glaucomatous eye which has been trephined or treated by iridencleisis. About 2 or 3 mm. from the limbus in the 11 and 2 o'clock meridia he makes incisions concentric with the limbus and through half the thickness of the cornea. Through the lips of these he then inserts a corneal suture. A corneal section is then made with a Graefe knife, which is brought out through the corneal incisions. The cataract is removed, preferably by the intra-capsular

technique. The author's diagrams show that on tying the corneal sutures the fistula made by the previous trephine or iridencleisis operation is kept open. He has operated on four patients using this technique. There were no complications and in each the glaucoma continued to be controlled. Two figures illustrate well the technique of this operation.

H. B. STALLARD.

IV.—MEDICAL OPHTHALMOLOGY

(1) **Simons, L. T. (Saint Paul, Minnesota).—Methyl-Alcohol amblyopia.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 446, 1942.

(1) **Simons** gives a historical survey of methyl alcohol blindness. He states that 10 c.c. is a toxic dose which will produce blindness and 100 c.c. will cause death. Methyl alcohol has a cumulative effect and may cause blindness in "tipplers" who at no time may have reached the toxic dose. Inhalation of methyl alcohol may cause poisoning, 0·2 per cent. in the air is sufficient for this. The prognosis is always bad and the incidence of visual recovery small. Formic acid, a product of oxidation of methyl alcohol is responsible for the visual disturbances, a toxic neuritis of the retro-bulbar part of the optic nerve is produced.

The author treated a case 17 days after the onset of methyl alcohol poisoning by a saturated solution of potassium iodide 30 minims daily and thiamin chloride (vitamin B_1) 2,400 international units a day by mouth. He does not claim a cure for this therapy as there was some visual improvement before it was started and he comments that the good result might have been the same regardless of treatment.

H. B. STALLARD.

(2) **Dvorak-Theobald, G. (Oak Park, Illinois).—Histologic eye findings in arachnodactyly.** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 1132, 1941.

(2) **Dvorak—Theobald** describes the case of a coloured child, aged 27 months, suffering from arachnodactyly. One eye was excised after death from an acute exanthema. The eye measured 33 mm. antero-posterior by 26·5 mm. horizontally and 26 mm. vertically. The surface distance between the cornea and optic nerve on the nasal side was 29 mm. and on the temporal side 45 mm. Structural defects were present at the filtration angle, incomplete separation between the iris and trabeculum; the iris was bowed backward; the dilator pupillae was absent and replaced by a homogeneous pink-staining substance; and the pigmentation of the posterior layer of the pars iridica retinae ceased half-way between

the pupillary margin of the iris and the base of the ciliary processes. In the ciliary body there was a great scarcity of circular fibres. The lens was ectopic.

The authors comment that structural disorders of the eye are present in more than 50 per cent. of cases of arachnodactyly. Arachnodactyly is hereditary and familial. The theories of origin of this disorder are discussed under the headings (1) congenital mesodermal dystrophy (2) hypophyseal (3) mixed theory of François. Six microphotographs illustrate this paper.

H. B. STALLARD.

V.—MISCELLANEOUS

(1) **Nordmann, J. (Strasbourg).—Tumour of the pars ciliaris retinae. (Les tumeurs de la rétine ciliaire).** *Ophthalmologica*, Vol. CII, p. 257, November, 1941.

(1) Nordmann gives a useful summary of the literature on the tumours of the pars ciliaris retinae and concludes that all such tumours consist of rows of cells and tubes of cells. Benign growths are either inflamed pseudo-tumours or Fuchs's epithelial tumours. These latter develop into cysts in the senile ciliary body. The malignant tumours also fall into two groups, depending upon whether embryonal or adult cells are the starting point. The embryonal type of tumours are neuro-epitheliomata or medullo-epitheliomata similar to those so frequently found in the central nervous system.

ARNOLD SORSBY.

(2) **Brückner, R. (Basle).—Cicatricial contraction of the conjunctiva following tissue damage in dyers. (Narbige Schrumpfung der Conjunctiva als Folge einer Gewerbeschädigung im Färbereiberuf).** *Ophthalmologica*, Vol. CII, p. 221, October, 1941.

(2) **Brückner** reports two cases of bilateral shrinkage of the conjunctiva in elderly dyers. He holds this to be an occupational disease induced by chemical vapours possibly aided by physiological changes in senescent tissues.

ARNOLD SORSBY.

(3) **Fischer, F. P. and Huysmans, J. H. B. M. (Utrecht).—The gas metabolism of the lens and vitreous. (Ueber den Gasstoffwechsel der Linse und des Glaskörpers).** *Ophthalmologica*, Vol. CII, p. 275, November, 1941.

(3) **Huysmans** and **Fischer** describe an apparatus for the measurement of the metabolism of explanted tissue and organs both

continuously and discontinuously. They show that the surviving explanted lens uses up oxygen and produces carbonic acid and has a respiration quotient of 1. The lens requires carbohydrates, which are partly oxydised, partly glycolised, and in part used for the production of vitamin C. The quantitative analysis of respiration, aerobic glycolysis and vitamin C formation showed that a carbohydrate residue remains, the use of which is still unknown.

The vitreous has no gas metabolism.

ARNOLD SORSBY.

(4) **Fischer, F. P. (Utrecht).—Senescent changes in the eye. (Ueber Altersveränderungen des Auges).** *Ophthalmologica*, Vol. CII, p. 226, October, 1941.

(4) Fischer classifies findings in over 2,200 patients aged 1 to 80 years and establishes frequency curves for arcus senilis, depigmentation of the iris stroma, pigmentation of the posterior surface of the cornea, depigmentation of the edge of the pupil and the pigment sheath of the iris and of lens opacities. All these senile changes increase directly with age in a regular curve, possessing all the characteristics of a saturation curve. The formula and importance of such conformity are discussed.

ARNOLD SORSBY.

(5) **Francois, J. (Charleroi).—Homonymous hemianopia in acute carbon monoxide poisoning. (Hémianopsie homonyme par intoxication oxycarbonée aiguë).** *Ophthalmologica*, Vol. CIII, p. 143, March, 1942.

(5) Francois reports a case of acute carbon monoxide poisoning in a woman aged 49 years. There was blindness with intact light reaction of the pupils. Vision slowly improved leaving right homonymous hemianopia. He holds that the underlying mechanism was asphyxia of the optic radiation with recovery on one side. A review of the literature is given from which it appears that two cases of homonymous hemianopia, one of binasal hemianopia and four of cortical blindness have been reported in carbon monoxide poisoning.

ARNOLD SORSBY.

(6) **McAlpine, P. T. and Berens, C. (New York).—Allergic dermatitis and blepharo-conjunctivitis caused by pantocaine.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 206, 1942.

(6) McAlpine and Berens describe 3 cases of allergic blepharo-conjunctivitis, and occupational dermatitis occurring in 4 ophthalmologists whose thumbs and fingers came into contact with swabs moisted in pantocaine after the instillation of this local anaesthetic into a patient's eye.

H. B. STALLARD.

(7) **Thomas, Maxwell (Dallas, Texas).—Knife injury.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 210, 1942.

(7) **Thomas** describes the case of a white boy, aged 16 years, who was stabbed in the left eye by a jack-knife. A photograph shows very graphically the knife entering the orbit through the plica semilunaris and a radiograph demonstrates it traversing both nares. Strong force was required to remove it under general anaesthesia. The eye and extra-ocular muscles were quite undamaged. Healing was uneventful.

H. B. STALLARD.

(8) **Curdy, R. J. (Kansas City, Missouri).—The influence of gravity on intra-ocular foreign bodies.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 208, 1942.

(8) **Curdy** stresses the importance of careful localization of intra-ocular foreign bodies in the lying position immediately before operation for extraction. In some cases it may be necessary to operate on the patient in the prone position so that gravity may make the foreign body more accessible. In such, radiographs taken with the patient's head in some other position will not be helpful when the foreign body has moved by gravity in the vitreous. He quotes a case of spontaneous extrusion of a foreign body through the lower part of the sclera several years after the eye injury.

H. B. STALLARD.

(9) **Lehrfeld, L. (Philadelphia).—Is enucleation indicated in early cases of intra-ocular malignant melanoma?** *Amer. Jl. Ophthal.*, Vol. XXV, p. 199, 1942.

(9) **Lehrfeld** comments on the doubtful value of excision of an eye for malignant melanoma of the uveal tract as regards longevity. He describes the clinical features of two males each aged 49 years. One patient had his eye removed when the vision was 5/12 in 1933 and died of metastases in March, 1941. The other had in 1930 a sightless eye containing a malignant melanoma and was suffering pain from the secondary glaucoma which this caused. He refused excision until November, 1940, when fungation of the growth into the orbit necessitated this operation. He died after an automobile accident in February, 1941. A supra-sellar metastasis was found at post mortem.

The author remarks that the spindle-celled types of malignant melanoma are slowly growing, that necrosis occurs in many cases (Terry and Johns, *Amer. Jl. Ophthal.*, Vol. XVIII, p. 903, 1935, found necrosis in 67 out of 94 tumours) and that early enucleation and exenteration does not prevent metastasis or death.

On the grounds that the tumour cells arise from neuro-ectodermal

cells and that these cells are destroyed by alcohol he puts forward a theory, at present unsubstantiated by clinical trial, that cases of early intra-ocular malignant melanoma should be treated by alcohol injections into the ciliary nerves.

[The author does not mention that some of these neoplasms are radio-sensitive but less so than glioma retinae and have been reduced in size and apparently checked in growth for several years. Diathermy has also been effective in destroying small growths either directly affecting the cells or by so damaging their blood supply that they are unable to live.—H. B. S.]

H. B. STALLARD.

(10) **Davis, W. T. and Neisenbach, A. E. (Washington, D.C.).—Ophthalmoscopic findings in draftees from the Washington D.C. Military Area.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 181, 1942.

(10) **Davis** and **Neisenbach** classify in their paper the ocular findings in 223 draftees referred to them from an intake of 13,280 men between November 1, 1940 and May 1, 1941. They comment on the low incidence of malingering in this group. Only 3 attempted to feign poor vision. Of the 223 men examined 39·9 per cent. were classified as 1A, a minimum vision of 20/400 without glasses corrected to 20/40 in either eye with glasses. 41·2 per cent. were placed in 1B, available for limited military service, and 18·3 per cent. were rejected for military service.

26 per cent. of the 223 were myopes and 43·5 had myopic astigmatism. The authors suggest that such an investigation of the eyes of the male youth of the nation will be of statistical value and may serve some useful purpose in arresting certain ocular diseases and preventing complications of these.

H. B. STALLARD.

(11) **Julianelle, L. A., Boots, R. H. and Harrison, G. H. (Saint Louis).—The treatment of staphylococcal infections of the eye by immunization with toxoid.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 431, 1942.

(11) **Julianelle, Boots** and **Harrison** studied the effects of immunization with toxoid on 30 patients suffering from chronic conjunctivitis. Pathogenic staphylococci were isolated from each patient's conjunctiva and tested by fermentation of mannite and extraction of type A carbohydrate. No other treatment was given these cases. Seventeen in addition showed superficial punctate keratitis, 3 had styes and 2 chalazia. By the end of immunization the serum showed a rise in antitoxin. At the end of the period of observation 10 had varying degrees of improvement and 20 gave no evidence of any change.

Of 22 patients examined 5 to 6 months after immunization 9 were about the same, 1 was improved and 12 were completely symptomless. The authors conclude that immunization with toxoid does not appear to be an effective method of treatment in staphylococeal eye infections.

H. B. STALLARD.

(12) **Doherty, W. B. (New York).—Some of the most important ocular and orbital wounds in war.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 135, 1942.

(12) **Doherty** quotes freely from Félix Lagrange's publication of war injuries of the orbit. He describes a routine method of examining these cases and gives a clinical report of nine patients, but not all of these were battle casualties. The paper is illustrated by radiographs and a fundus painting in colour.

H. B. STALLARD.

(13) **Haldimann, C. (Berne).—Corneal and conjunctival changes in Schaumann's disease. (Hornhaut- und bindehautveränderungen bei Boeckscher Krankheit).** *Ophthalmologica*, Vol. CII, p. 138, September, 1941.

(13) **Haldimann** describes two characteristic cases of Schaumann's disease (Boeck-Besnier Disease; benign Lymphogranulomatosis), showing calcareous depositions on both the cornea and conjunctiva. The blood calcium in both cases was raised and the author believes that this is not an uncommon occurrence in this affection.

ARNOLD SORSBY.

(14) **Francois, J. (Charleroi).—Rhabdomyosarcoma of the orbit. (Rhabdomyosarcom de l'Orbite).** *Ophthalmologica*, Vol. CII, p. 129, September, 1941.

(14) In addition to the 13 cases of rhabdomyosarcoma in ophthalmological literature, the author describes a case observed by himself, in a woman, aged 61 years. After initial paresis of the abducens the complete features of orbital tumour developed within a month. Histological examination after exenteration of the orbit showed cells with thick, definitely enlarged nucleus and eosinophile plasma with distinct long and cross striping. Four weeks later death occurred from lung metastasis. The interest in this case lies in the fact that it shows the extremely malignant character of this affection, which is generally present at birth, but which may develop later in life, as in this patient.

ARNOLD SORSBY.

(15) **De Ruyter, J. (The Hague).—Aetiology of senile cataract. (Ursachen des Altersstares).** *Ophthalmologica*, Vol. CII, pp. 93-103; and 146-163, August and September, 1941.

(15) De Ruyter's article, whilst not containing any personal observations, is a useful survey of the recent literature on the pathogenesis of cataract. The article contains 81 references.

ARNOLD SORSBY.

CORRESPONDENCE

PRESCRIBING OF SPECTACLE LENSES

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—The attention of the Council of British Ophthalmologists has been drawn to the difficulty which the Services and the Ministry of Health are experiencing in obtaining spectacle lenses to supply their urgent needs, and it has been suggested that the situation could be helped by a simplification of the prescriptions supplied by civilian ophthalmic surgeons.

The Council has considered the question and makes the following recommendations:—

1. That the prescribing of lens powers in steps less than 0·25 D. (which is to say, lens powers commonly referred to as "eighth sights," *e.g.*, 0·87 D.) be discontinued.
2. That the prescribing of prisms be reduced to a minimum.
3. That in the higher ranges, say above 4 dioptres, the prescribing of lens power in steps less than 0·5 D. be reduced to a minimum.

The above recommendations were adopted by the Army some time ago, and the Council is assured that their wide adoption in civilian practice would materially aid the situation.

I have the honour to be, Sirs,

Your obedient servant,

FRANK W. LAW.

Honorary Secretary, Council of British Ophthalmologists.

36, DEVONSHIRE PLACE,
LONDON, W.1

March 23, 1943

NOTES

Oxford Ophthalmological Congress

DR. BERNARD SAMUELS, of New York, has accepted the post of Deputy Master of the Oxford Ophthalmological Congress in the place of Mr. P. H. Adams, who has resigned after nearly 30 years in that office.

* * * *

North of England Ophthalmological Society

AT the Annual Meeting of the North of England Ophthalmological Society the following officers were elected for the session 1943-44. *President*, H. Mather (Liverpool); *Vice-President*, C. F. Eminson (Doncaster); *Members of Council*, J. A. McCann (Liverpool) and Miss G. B. Leigh (Sunderland); *Treasurer*, W. H. Kiep (Bradford); *Secretary*, W. M. Muirhead (Sheffield).

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Royal Society of Medicine

THE meetings of the Section of Ophthalmology during the session 1942-43 will be held as follows:—

Friday, June 11, at 5.0 p.m.

Cases and specimens will be shown half-an-hour before the advertised times of the meetings.

* * * *

International College of Surgeons

THE fourth International Assembly of the International College of Surgeons will be held on June 14, 15, 16, at the Waldorf Astoria Hotel, New York. Dr. F. H. Albee is President, Dr. Thorek, the executive Secretary. The programme will be devoted to war surgery and rehabilitation.

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Special Notice

WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.